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TRAUMATIC AND ORTHOPAEDIC SURGERY

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EDITORIALS

It is said that the casual visitor to the university campus can often identify with little difficulty the budding lawyer, engineer and doctor. Each has usually begun, even before graduation, to reflect the pattern of the personality stereotype that will be his in later years. Considering the specialized training that such a student receives, and considering the specialized nature of the professional service he will render after graduation, it is neither remarkable nor undesirable that he should acquire some share of the characteristics traditionally attributed to the professional man.

It is nevertheless true that, in the moulding of character and personality along such strict and inflexible lines, there are definite hazards, both to the individual concerned and to those to whom his services are dedicated. It happens not infrequently that the physician is so involved in his professional duties that he has not the time to relax properly and to find enjoyment in day-to-day living. Although such a situation is a tragic one, the tragedy is personal and does not necessarily carry over into the lives of patients.

A more sinister hazard lies in the process of acquisition of the confidence and self-assurance that are, in proper degree, so necessary in the successful handling of the patient. There is never any justification for a failure to maintain some spark of intellectual humility. It both restricts the physician to those measures of diagnosis and treatment of which he feels properly capable, and also keeps before him the realization that there are an infinite number of things yet to be learned.

It is in his student days that the individual has the chance to build such a constructive attitude, and there can be no doubt that many students fail utterly to keep an open mind, to see a problem from many sides and to know the value of emphasis placed upon fundamental principles. For the individual who feels so insecure that he dare not put aside his texts long enough to take some positive part in the society around him, there can only be pity; and for the individual who feels that he need not study because he has learned enough, there can only be contempt.

If there remains somehow inherent in the professional man's personality stereotype a proper zest for living and sufficient zest for learning, then the adherence of any individual to such a pattern must remain above criticism.

W.H.M.

The P.O.T. Contribution

This issue marks a fuller degree of liaison among the students of the Faculty of Medicine: that is, between the medical students and the students in the course in Physical and Occupational Therapy. The article "Post-Traumatic Rehabilitation" was selected by the teaching staff and the editors from several contributions by girls in this course. One hundred and twenty-eight P.O.T. students are new subscribers to the Journal this year. We would like to acknowledge this unusual and heartening enthusiasm.

CHEST TRAUMA

S. SHORE, 5T5

WITH the ever increasing incidence of automobile accidents, not to mention other hazards, the necessity for prompt and proper treatment of chest injuries has become a much more common problem.

Anatomy and Physiology

The lungs may be compared to two balloons having a common neck and each contained in a pleural cavity which is only a potential space. The mediastinum with its contents lies between the two lungs and its position is maintained by the equality of pressure in the two pleural cavities. Both the lungs and mediastinum are enclosed partly by a rigid framework of ribs, sternum and vertebrae and partly by soft structures, notably the diaphragm and intercostal muscles. On inspiration, the thoracic cage enlarges, resulting in an increased negative pressure in the pleural cavities. Thus a suction effect is exerted upon both the lungs and the thoracic cage, with air being drawn into the lungs. The thoracic cage remains relatively unaffected because of the resistance produced by its rigid components. On expiration, with decrease in size of the thoracic cage, the negative suction pressure is decreased and elastic recoil of the lungs forces air out.

The pulmonary air passages are kept clear of foreign material and excess secretion by a combination of ciliary movements, a thin mucus coat lining the tracheobronchial tree and the cough reflex.

The pulmonary blood pressure is approximately one-sixth of the systemic blood pressure, the pulmonary arterioles being short, wide, thin-walled and with an enormous capillary bed. These factors result in a very small peripheral resistance to the output of the right side of the heart, Thus pressure changes inside the thorax appreciably affect blood flow, negative pressure encouraging flow toward the great veins and heart.

Respiration is under voluntary and involuntary control. Its regulation is carried out by the integrated action of muscular, nervous and chemical mechanisms, which are under control of the respiratory centre in the medulla.

Emergency and General Treatment

In any chest injury, a cursory examination should be carried out, the level of consciousness determined, and pain relieved by a narcotic such as 25-100 mg. of Demerol I.V. or I.M., which has a minimal depressive effect on the respiratory centre. All serious chest injuries should be quickly transported to hospital in the erect or semi-erect position, with the injured side dependent. Thus respiration is aided by lowering of the diaphragm, and flooding of the sound side by blood and secretion is less likely to occur. In those cases where cardiorespiratory function is endangered, immediate treatment must be commenced. Lacerations causing sucking wounds of the chest must be promptly covered with a tight dressing to prevent free entry and exit of air between the external environment and the pleural cavity. In removing fluid as in a hemothorax, depending on the level of dullness to percussion, the needle is usually inserted below the tip of the scapula or above the 8th costal cartilage in the mid-clavicular line. These conditions will be discussed in more detail later.

Where indicated, tracheotomy, by incision through the 3rd cartilage of the trachea, is a life-saving procedure to maintain a patent airway. One must always be aware of possible multiple injuries elsewhere in the body.

Upon arrival at the hospital another rapid examination should be made and where the injury is serious the following procedures must be considered.

SHOCK Blood and plasma should be supplied but saline avoided since it increases the danger of pulmonary edema.

Where indicated by marked difficulty in respiration or cyanosis, oxygen is best administered by nasal catheter to facilitate nursing care and examination of the patient.

The relief of pain is essential since pain inhibits deep breathing and the cough reflex. This results in rapid, shallow breathing which may lead to anoxia and cynosis due to inadequate ventilation. With depression of cough reflex, there is a failure to maintain the air passages clear of secretions or blood This may be severe enough to cause obstruction and absorption collapse of lung. Alleviation of pain may be obtained by intercostal nerve block, infiltrating immediately below the involved ribs with 1% procaine, or by the injection of Demerol.

PATENT AIRWAY In order to maintain a patent airway, the patient must be encouraged to breathe deeply and to cough. Intratracheal aspiration by catheter or bronchoscopy under local anaesthetic can be done at the bedside. In severe injuries, tracheotomy must be considered to provide ready access in draining secretions and to lessen the effort of breathing.

LUNG RE-EXPANSION Lung collapse results from air, blood, chyle, effusion, or abdominal viscera in the pleural cavity with subsequent respiratory embarrassment. An accurate diagnosis should be made and treatment rapidly instituted.

Adynamic ileus following chest trauma leads to abdominal distension which will elevate the diaphragm and cause further respiratory difficulty. This should be relieved by intestinal decompression.

ANTIBIOTICS Antibiotics should be given prophylactically to prevent subsequent infection.

Once the patient is out of immediate danger, a more extensive physical examination and radiology may be carried out. Do not send a patient for X-ray until all emergency treatment has been completed since the delay may cost the patient his life.

* * *

Thoracic Trauma may be divided into 3 groups:—

1. Non-penetrating injuries
2. Penetrating injuries
3. Blast injuries.

The presence of trauma to thoracic contents and diaphragm may be difficult to diagnose when more obvious injuries such as rib fractures are present but must always be kept in mind.

Non-Penetrating Injuries

RIB FRACTURES Rib fractures may be caused by direct blows, crushing injuries, and occasionally by muscular strain, severe coughing and sneezing. The patient often presents with chest pain aggravated by respiration, with maximum tenderness and perhaps crepitus over the fracture site. There may be marked voluntary splinting of the abdominal muscles on the affected site. Interstitial emphysema only occurs if the pleurae and lungs are ruptured. Final diagnosis may be made radiologically. The patient should be closely followed because of possible complications such as pneumothorax or hemothorax.

Treatment consists of immobilizing

the fractured ribs by strapping with 2" adhesive applied in full expiration, extending at least 3" on the sound side of the chest, front and back. Alternatives are a firm support or binder.

Pain may be relieved by infiltrating the intercostal nerves with procaine, including a rib above and one below the fracture.

FRACTURES OF THE STERNUM AND COSTAL CARTILAGES Since *sternal fractures* are usually incomplete or undisplaced, chest wall stability is not endangered and treatment is simply local procaine-in-oil injection. However, injury to thoracic organs is common and must be watched for. If the fracture is complete with displacement, the treatment of choice is open reduction and wire fixation. Other possible methods are strapping, hyperextension plaster mold, or fixation by suture.

The *fracture of costal cartilages* may be seen in younger adults and the diagnosis is made by palpation and the occurrence of a typical click associated with movement of the chest. Unless calcification of the cartilage has occurred, radiology reveals nothing, and treatment is similar to that for sternal fractures.

STOVE-IN CHEST In this condition *multiple ribs or cartilages* are fractured in two or more places, resulting in paradoxical movement of the *detached chest wall* segment. That is, the segment is drawn inward on respiration due to increased negative intrapleural pressure and forced outward on expiration due to decreased negative intrapleural pressure. Thus pulmonary ventilation is impaired on the injured side, and if the detached segment is large, the lung on the involved side may actually become smaller on inspiration. On expiration, the lung on the sound side, by retracting, forces deoxygenated air back into the other lung. As a result of the inequality of pressures between the pleural cavities, mediastinal flutter occurs with the phases of respiration causing serious embarrassment to the heart and great vessels. In addition, the cough reflex is inhibited and reflex motor and secretory stimulation of the bronchial tree occurs.

The main symptom is dyspnoea, often accompanied by cyanosis.

Serious immediate complications may result such as mediastinal emphysema, tension pneumothorax, hemothorax, or injury to the heart and great vessels. Later complications include empyema, lung abscess and mediastinitis.

Treatment includes maintaining a clear airway, and tracheotomy should be advocated if there is progressive mediastinal emphysema, characterized by crackling sounds on auscultation of the heart.

Stabilization of the chest wall is essential. Firm strapping over a pad or sandbags is beneficial if the injury is unilateral. However, in bilateral injuries, this method further decreases the already impaired respiratory reserve. A more effective method is skeletal traction by a tenaculum in the sternum or a durable steel wire from the ribs with traction over a pulley. Another method is open reduction of the fractured ribs, employing bone-graft supports if indicated.

Penetrating Injuries

These may be internal from comminuted rib fractures, or external from stab or gunshot wounds.

LACERATIONS Lacerations of the chest wall which are superficial should be cleaned and sutured, and the patient carefully followed for possible bleeding.

MISSILES *High velocity* missiles passing completely through the chest cause little damage to the chest wall, but may

result in extensive intrathoracic trauma. After shock has been treated, the wounds are cleaned, debrided and closed. The patient is then kept under close surveillance for complications due to injured thoracic contents.

Serious injuries to the thoracic cage are more common from *low velocity* or large irregular missiles. Large *sucking wounds* may result in free communication between the pleural cavity and the external environment. There is consequent lung collapse, mediastinal flutter, impaired cardiovascular function and stagnation of air in the bronchial tree. Immediate closure of the wound with dressings andtight packing is essential. On arrival at hospital, repair of injury to underlying organs may be undertaken and the chest closed, providing the patient's general condition is satisfactory.By leaving the needle or catheter inserted in the pleural cavity, conected to a closed under-water seal, closed drainage is maintained. This keeps the pleural cavity dry and encourages rapid re-expansion of the lung.

Retained missiles in the chest should be removed, as a rule, after accurate pre-operative localization once the patient is out of immediate danger. Possible complications are haemoptysis, empyema, and abscess formation.

TENSION PNEUMOTHORAX This condition arises when the lung is perforated with the lacerated lung exerting a *ball valve* effect so that air enters the pleural cavity on inspiration, but cannot escape during expiration. The increase in positive intrapleural pressure results in progressive collapse of the affected lung and mediastinal shift to the opposite side. The patient becomes increasingly dyspnoeic, often cynosed, and examination reveals hyperresonance and decreased breath sounds over the involved side. There may be an accompanying haemothorax resulting in further lung compression from the accumulation of bloody fluid in the pleural cavity.

Treatment consists of immediate thoracentesis followed by continous drainage with a closed underwater seal until spontaneous closure of the leak occurs, and the lung begins to expand.

The possibility of a bilateral pneumothorax or a contra-coup pneumothorax must always be kept in mind.

HAEMOTHORAX Bleeding most commonly arises from the lung and tends to stop spontaneously due to the low pulmonary blood pressure. Haemotysis occurs and daily aspiration of the haemothorax is indicated until less than 50 cc of blood is obtained.

Bleeding from intercostal vessels is a more acute emergency due to the high systemic pressure, and often necessitates a direct attack operatively to stop bleeding.

The patient is often pale, restless, and thirsty. Examination reveals a falling blood pressure, a weak thready pulse, dyspnoea and cyanosis. There is flatness on percussion and decreased or absent breath sounds on the affected side, and shifting of the mediastinum to the opposite side.

Since blood irritates the pleurae, there is commonly a secondary pleural effusion after the first 24 hours, which may rapidly lead to dangerous decompression symptoms.

Delayed or inadequate treatment may result in multiple adhesions or the formation of a fibrinous coat from clotted blood covering the pleurae, thus preventing free movement of the lungs and interfering with respiratory function. The use of Tryptar or Varidase injected into the pleural cavity may break down these adhesions by their proteolytic action, but care must be taken not to leave these agents in the pleural cavity too long for fermentative destruction of lung

parenchyma may result. If this fails, decortication by opening the pleural cavity, removing the adhesions or fibrinous casts, and fully expanding the lung under direct vision may be indicated.

ABDOMINO-THORACIC WOUNDS When bilateral abdominal rigidity occurs and persists after effective treatment of the chest injury, there may be concomitant abdominal haemorrhage or perforation. Careful exploration either through the thoracic wound or through a separate abdominal wound, particularly in penetrating wounds, is indicated.

Blast Injuries

Explosions produce a momentary marked increase in air pressure, immediately followed by a negative or suction wave, the former causing the most damage. Rupture of vessels and alveoli in the lungs with bloody exudate seeping into the bronchi results in haemoptysis. There is also a tamponade effect on the heart so that blood is squeezed out of the thin walled auricles into the pulmonary veins and vena cava.

There may be a latent period before signs of shock, dyspnoea, cough and haemoptysis appear.

Treatment consists of complete bed rest, the administration of oxygen, the maintenance of a clear airway, and attention to any wounds or complications.

Injury to Thoracic Contents

LUNG Parenchymal lacerations of the lung usually heal spontaneously. Occasionally, suturing of large lacerations, or lobectomy where there is gross destruction of a lobe, is indicated.

Tears in a main bronchus or the trachea require early operative treatment. Rapidly increasing mediastinal emphysema suggests the diagnosis, and confirmation is made by bronchoscopy.

OESOPHAGUS Lacerations of the oesophagus from penetrating injuries present a picture of sudden, severe mediastinal or lower thoracic pain. Shock, mediastinal emphysema and pneumothorax may occur.

Diagnosis may be made by barium swallow, showing accumulation in the chest. There is usually co-existent chest injury and early operative repair is indicated.

DIAPHRAGM Rupture of the diaphragm from a compression injury may be suspected when there is progressive shortness of breath or increasing abdominal symptoms. The stomach is displaced into the thoracic cavity, and the diagnosis is made by barium swallow and chest X-ray. Since there are usually multiple injuries present, the diagnosis is often difficult.

HEART Contusions of the heart from compression injuries may cause sudden death, or result in myocardial insufficiency.

Wounds of the pericardium and heart are generally due to penetrating injuries. In those cases which survive, cardiac tamponade often results with haemopericardium and secondary pericardial effusion.

The patient is in shock, and shows an increased area of cardiac dullness on percussion and distant heart sounds.

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FAT EMBOLISM AS A COMPLICATION OF FRACTURES

W. H. McCRAE, 5T5

IN the condition known as fat embolism, globules of liquid fat are found to produce vascular occlusion, especially of arterioles and capillaries. Characteristic clinical and pathological pictures may be produced, which are fundamentally attributable to this occlusion, and to subsequent local reaction, including haemorrhage.

Although mention was made of fat emboli in the literature as early as 1669 (by Lower), it is acknowledged generally that the first article relating the phenomenon to human disease is that of Zenker in 1862. He described fat emboli observed in the pulmonary capillaries of a railroad worker who had suffered a severe crushing injury. Since that time, many cases have been reported (over 650 cases by 1951), but differences of opinion concerning the importance of fat embolism as a clinical entity have always persisted. In recent years, particularly since the second World War, there has been much agreement that in the wake of gross mangling and some lesser degrees of trauma, especially trauma wherein long bone fracture is involved, fat embolism should be suspected as a cause of delayed recovery or of death itself. Alldred believes that it "must be regarded today as one of the commonest and most serious complications of a fracture".

Incidence

Fat embolism commonly follows fractures, crushing injuries and surgical manipulations of long bones, most notably the femur and tibia. In one series, 91% of cases had such a history. The condition may also follow trauma to abdominal viscera, severe burns, damage to fatty liver tissue, and many other disease states. Although these other causes will not be discussed here, they ought not to be forgotten.

Fat embolism is more common in males than in females, especially in young adults and those engaged in manual tasks which might lead to fractures or crushes. Fatty emboli are cast free in everyone's bloodstream from time to time, but it is only occasionally that clinical symptoms result.

Authors who emphasize meticulous autopsy studies and sputum and urine examinations for fat generally report a higher incidence of fat embolism than those who stress the detection of clinical signs. In 1953, Glas reported that, on the basis of finding free fat in the urine, some 52% of 109 accident cases consecutively admitted to hospital had evident fat embolism. The phenomenon was seen in

59% of fracture cases
57% of burns
25% of contusions and lacerations.

Recent studies of men killed in Korea are said by Glas to show that about 33% had fat embolism in significant degree and that some four-tenths of this 33% had their deaths attributed to this cause.

The records of the Department of Pathology in the Banting Institute, Toronto, show that, on the average, one death each year is set down as fairly clearly attributable to fat embolism.

Mechanism of Production

SOURCE OF THE FAT There have been two chief theories advanced for embolus production. A few workers, notably

Lehman, put forth over 20 years ago the opinion that there must be some disturbance of the physiologic emulsion in plasma of fatty materials by agents both physical and chemical, and that the quantities of fat "thrown down" are then sufficient to evoke the clinical picture which is described below. They contended that no one had shown that fat freed from marrow was sufficient in quantity to produce all the symptoms, and that the explanation for fat entry into the bloodstream was unsound.

The opponents of this "in situ" view believe that the fat is introduced into the venous circulation typically from the fat-rich marrow of fractured long bones. Since this latter proposition enjoys the support of most current evidence, it will be developed here without further reference to the opposing theory.

NATURE OF THE EMBOLI The more popular view states that after some disruptive injury, such as a fracture, some of the fat cells in the marrow in the injured area are broken down and torn free. The result is a number of globules of almost pure fat, in various shapes and sizes but often less than 15 mu in diameter. It is said that 1 cc. of fat will form about 6,000,000 droplets 15 mu in diameter. The average capillary width is 5-10 mu. The final shape and disposition of emboli at their site of lodging are determined by such factors as vessel calibre, surface tension at fat-plasma interfaces and circulatory pressure. Definitive globule size at the final site may reach 1 cm. diameter. The massive oil-water interfaces are blamed for the behaviour of these emboli as solid obstacles in the bloodstream.

PATHS TRAVELLED Marrow pressure is normally intermediate between arterial and venous pressures (Bloomenthal 1952). A rise in the effective marrow pressure, occasioned by haemorrhage and other exudation into a relatively non-expansible space, may suffice to introduce fat globules into the lumina of small venous channels kept patent by the bony framework around them. Once in the venous circulation, the fat is carried toward the right side of the heart, and is thought to navigate it without incident.

The lung capillaries and arterioles may then become swiftly loaded with fat globules. Although vascular spasm is not thought to occur in these receptor areas, Harris and others have suggested that pathological changes here are in some measure attributable to the decomposition of fat globules by tissue lipases, with the release of irritating fatty acids. Scuderi has blamed oleic acid in particular.

The fat reaches pulmonary vessels within minutes of release. Within a few hours, fat may be present in the alveoli, and hence eventually appear in the sputum. Some of the fat remains intravascular and emerges in the efferent lung vessels to traverse the left side of the heart and be distributed to the peripheral regions of the body.

Microscopic Pathology

Greatest attention has been given the introduction of fatty emboli into the vessels of the lungs, nervous system, kidneys, skin and heart.

LUNGS Studies of autopsy specimens (Wyatt and Khoo 1950) suggest that cellular reaction to intravascular fat droplets is at first slight. Within 24 hours there are signs of local oedema and haemorrhage. The first cells to appear are neutrophiles, and the picture is roughly that of an early pneumonia, apparently constituting an acute foreign body reaction. Fat which reaches the alveoli is engulfed in part by mononuclear phagocytes, and after about 36 hours, fat may appear in the sputum. One may sum up

the pulmonary changes as a sequence of occlusion of small vessels, local anoxia, increased capillary permeability, oedema and infiltration of inflammatory cells.

CENTRAL NERVOUS SYSTEM The white matter may reveal petechial haemorrhages in areas supplied by small end-arteries. These are a kind of microinfarct, with small areas of necrosis and peri-capillary extravasation of blood. Sub-cortex, internal capsule, midbrain and lower brain stem areas may be involved.

The gray matter does not customarily revel petechiae although alteration of function may be evident clinically.

KIDNEYS There are to date no clear-cut examples of serious renal damage due to fat embolism. Fat globules are found often in the lumina of glomerular vessels, but seem not to be associated with any disruptive changes nor with easily recognizable cellular reaction.

SKIN Petechial haemorrhages may appear over the trunk, abdomen, and in the conjunctivae, and are rarely found on the limbs. The typical neck-and shoulders distribution of petechiae is attributed by some to the ease with which the thyrocervical trunk may distribute emboli in the horizontal patient.

HEART Sections from at least three fatal cases on record at the Banting Institute show fat embolism in the capillaries of the heart muscle in varying degrees of severity, although haemorrhages around involved vessels are not noticeable in these particular sections.

One of these cases, that of an elderly female, is remarkable in that the patient died only three hours after fractures of tibia and femur had occurred. The Kodachromes show masses of intracapillary fat which stains well with Scharlach R. Death occurred too soon after fat embolism for the heart muscle itself to show any changes.

To summarize the findings on microscopic study of affected tissues, it is worthwhile to note that there is in each case, (excepting kidney) a general tendency to small-scale haemorrhage in the areas around occluded vessels. Also attendant upon occlusion are other changes which follow circulatory impairment, including congestion, oedema, cell death and cellular infiltrations.

Clinical Picture

Signs and symptoms may appear within a few hours after the injury, but the onset following fracture is commonly in 36 to 48 hours.

Following this "quiet period" there is a characteristic pattern of signs and symptoms which reflect directly the manner in which the emboli distribute themselves from their site of origin. Thus in the full-blown case there are symptoms of a pulmonary disorder first, followed sooner or later by evidence of involvement of the nervous system, skin and possibly other areas, via widespread arterial embolism.

A general change in the patient's condition is the first manifestation of fat embolism. There may be a restlessness and a rise in the pulse rate. The blood pressure commonly remains unchanged, but the body temperature may rise three degrees or more. The fever is said by some authors to be an inconstant finding, and may be attributable to one or more of:

1. Cerebral involvement by emboli.
2. Infection at the fracture site.
3. Secondary pneumonia.

Dyspnoea, chest pain, and cyanosis suggest pulmonary involvement. Moist chest sounds with patchy areas of dullness suggestive of consolidation are possible findings at physical examination. A cough and sputum may be present. Blood is sometimes found to tinge the sputum.

The appearance of a snowstorm phenomenon in the chest X-ray is striking and greatly aids diagnosis when it is present. However, this finding is not to be regularly expected.

Signs of involvement of the central nervous system may be generalized or have localizing value. Evidence of cortical involvement may range from restlessness or slight personality change to drowsiness, coma or mania. Cases showing petit mal attacks, a Jacksonian epileptic picture, and convulsions have been described. Urinary incontinence may be a sequel to damage to the appropriate nerve tracts.

Damage to the internal capsule can produce limb spasticity or hemiplegia.

Midbrain damage may be associated with pupillary changes, either in size or in reaction to light.

The affected brain stem commonly contains petechial haemorrhages, and embolic interference with temperature regulating centres may explain increases in body temperature.

A bright petechial rash often appears over the neck and upper chest, along with the appearance of other embolic manifestations. The rash begins to fade within 48 hours but it may remain visible for several days thereafter. It is not always confined to the neck and chest.

The conjunctivae may share in the display of petechial markings. The observation of fat globules in the retinal vessels is stressed by some authors but denied by others.

In fatal cases, the progression of symptoms may be accelerated. Occasionally, there is no symptom-free interval at all and the usual clinical pattern of events is not seen. The following is a resume of such a case taken from the records of the Department of Pathology, Banting Institute. (From a summary by Dr. J. H. Barrie)

A/220/50
Anatomical diagnosis: Multiple fractures.
Fat embolism.

This 27-year-old airman crashed from a low level into a lake, was thrown clear and was retrieved in a sufficient state of conseiousness to ask about the fate of his companion. He was taken at once to the hospital, where he lapsed into coma, became cyanosed, and died all within the spaee of three hours after the crash. Autopsy revealed multiple fractures, none of which should have been fatal. There was a large quantity of marrow fat lying free in a femoral fracture site, and there was microscopic evidence of massive fat embolism of the lungs, with lesser involvement of kidneys and brain. The left ventricle showed subendocardial haemorrhages.

Diagnosis

The possibility of fat embolism should never be ignored in accident cases, especially those wherein fractures are sustained. Alldred considers the following mnemonic useful:

Second hour: shock
Second day: fat embolism
Second week: pulmonary embolism

The detection of the clinical picture seems to be the cardinal means of diagnosis. Other aids include X-ray studies of the lung fields, investigation of the fundi for fat in the retinal vessels, and a search for fat in sputum and urine samples.

Some workers place high diagnostic value upon the demonstration of fat in urine specimens taken as 24-hour samples for several days consecutively after admission. Urinary incontinence developing about the second or third day may indicate damage to the nervous system.

Haematuria following traumatic renal infarction has not been attributed to fat embolism but is considered merely to coexist with the fat embolism in those cases displaying both findings.

It is worthwhile to state that the mere fact that a patient is obese does not ap-

parently increase his chances of suffering from fat embolism. Neither does the severity of the injuries parallel the incidence of this condition. It is true, however, that the severity of symptoms seems to vary directly as the amount of fat released.

Those interested in EEG findings in cases of fat embolism are referred to Kershman and Haddad.

Since frozen tissue sections, with the use of special stains, like Scharlach R and Sudan, or the use of osmic acid are often required to demonstrate fat emboli, the pathologist may sometimes miss the diagnosis unless he is given reason to suspect fat embolism from the circumstances of the death.

Course and Prognosis

In milder cases the physical signs are transient and may disappear in twelve hours or thereabouts after onset. Serious paralytic changes may persist for many weeks or months, but in the vast majority of cases, even those with some cerebral involvement, recovery may occur in 7 to 10 days. Eventual complete recovery is the rule, except in those cases wherein deep coma, Cheyne-Stokes breathing, and other evidences of profound nervous system involvement are seen. In the latter instances death is more likely. Occasional permanent damage may take the form of hemiplegia or mental defect.

Treatment

There is no specific treatment for fat embolism at this time. Early immobilization of fractures is held worthwhile in reducing the dissemination of emboli. Dyspnoea and cyanosis may be combatted with oxygen, and infection of lungs or fracture site with an appropriate antibiotic. Beyond that, one may only ensure that sensible supportive measures are taken. Indeed, since most patients with fat embolism do recover, the importance cannot be minimized of attention paid to fluid balance, bladder and bowel function, and to the early restoration of motor function.

Attempts to introduce into the bloodstream detergents capable of emulsifying the fat globules without damaging or irritating the body tissues have so far been unsuccessful.

Summary

Fat embolism has been known for many years but has lately received wider attention, especially as a complication of fractures of long bones in young adults. It is currently held that fat globules from the fracture site perfuse veins, the right side of the heart, the lungs, the left side of the heart, the brain, and peripheral tissues to produce a clinical picture which is essentially that of a pulmonary disorder coupled with widespread arterial embolism. The fat obstructs capillaries and arterioles to produce oedema or microinfarcts by local anoxia, and may possibly damage tissue by partial reversion to fatty acids. Recognition is usually by means of the clinical picture, with chest X-ray and urine fat studies as aids. Treatment at present is non-specific. Prophylaxis by early fracture immobilization is stressed.

Acknowledgement

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CLINICAL DIAGNOSIS OF SHOULDER INJURIES

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NO attempt will be made here to describe or classify shoulder injuries in any formal way, and little will be said about fractures, or about treatment. It is desired rather to limit discussion to clinical aspects of diagnosis, and in particular it is hoped to show that diagnostic thinking can be simplified if it is based upon a clear history of details of the manner of injury, interpreted in terms of direct effect on the anatomical structure and function of the shoulder.

ATLAS AND VENUS

The shoulder has been symbolic of strength from ancient times. Atlas carried the world on his shoulders; and we speak of "putting a shoulder to the wheel", and of "shouldering a load". From an anatomical standpoint however, the shoulder is more remarkable for its wide range of movement, evolved so well to serve the prehensile function of the upper extremity. But because of its wide mobility, it lacks something in stability, and so is more susceptible to injury.

SCAPULO-THORACIC STABILITY

Shoulder movement is a summation of movement of the shoulder girdle on the chest wall (scapulo-thoracic movement) and of movement of the gleno-humeral joint. The scapulo-thoracic component has lesser range, it is controlled and stabilized by powerful muscles, and it rarely would be affected by injury were it not that its function is modified by the presence of the clavicular strut which pivots at its lateral end on the scapula (acromion), and at its medial end on the trunk (sternum), maintaining a fixed distance between them. The clavicle and its articulations thus share in all movements of the shoulder girdle on the trunk, and form a weak link in the scapulo-thoracic system. The clavicle is prone to fracture, and its articulations, particularly the lateral one, to sprain or dislocation.

GLENO-HUMERAL WEAKNESS

The gleno-humeral joint contributes only about two-thirds of total shoulder movement yet its range still is greater than that of any other joint of the body; this being permitted by the low ratio of the articular area of the glenoid to that of the humerus, and by the laxity of the capsule of the joint. Stability depends greatly on the muscles that control its movement, and on the sheltering

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coracoacromial arch. The arch is separated from the musculo-tendinous cuff of the shoulder by the sub-deltoid or subacromial bursa, and is of such importance in shoulder joint function that it has been suggested it be called the "upper humeral joint".

THE RECOIL MECHANISM

The shoulder joint is protected also by the floating or gliding type of suspension of the shoulder girdle on the chest wall. The pelvic girdle is fixed to the trunk and forces transmitted along the axis of the fmeur meet the full impact of the body weight at the acetabulum. Because the shoulder girdle is suspended by powerful flat muscles, however, a force transmitted along the axis of the humerus does not meet full resistance of the body weight at the glenoid, but is taken up gradually by the hydraulic-like action of the suspending muscles which permits the whole shoulder girdle to recoil. Indeed, much of the force is not transmitted through the shoulder joint mechanism at all, but is taken up by muscles which pass directly from the humerus to the trunk. When one falls forward on the hands, the pectoralis major muscles take a large part of the load thus protecting the shoulder joint mechanism, and when one falls backward, the latissimus dorsi muscles have similar function.

A CLEAR HISTORY MAY BE THE CLUE

The shoulder, because of its location and function, is peculiarly subject to injury. The upper extremity is called upon instinctively to protect the body against injury, whether to parry a blow or to protect the body in falling, and if the intrinsic strength of any component part is overcome, injury results, whether to muscle, bone, capsule or tendon. In making a diagnosis of injury, much help can be derived if one obtains a clear history of the events of the injury, the nature and intensity of the acting force, its direction, and the manner of reception of the force by the body. One then can postulate the structure liable to injury, and the type of injury that will result. This is particularly of value in diagnosis of soft tissue injuries where X-rays offer no visual help.

INTO THE BOARDS

There are many common examples of direct injury about the shoulder. A blow against the outer aspect of the shoulder as when a hockey player is driven into the boards, is transmitted in large part through the clavicle, and if the force is excessive either the acromio-clavicular joint is sprained or dislocated or the clavicle is fractured. Injury of the acromio-clavicular joint is confirmed by finding of local tenderness, swelling and displacement.

ANTERIOR DISLOCATION IN YOUNG AND OLD

A fall with the arm outstretched which causes the arm to be carried upward and backward in external rotation beyond its normal range, pivots the neck of the humerus on the margin of the acromion, levers the head of the humerus against the antero-inferior capsule of the joint, and produces dislocation of the humeral head over the brim of the glenoid at its antero-inferior margin. Tautness of the capsule and ligaments of the normal joint is sufficient to prevent dislocation, so that a lesion of the capsule almost invariably occurs. Most commonly the glenoid labrum tears from the glenoid rim, but the capsule may tear from the glenoid labrum or there may be an intrinsic tear in the capsule itself. This establishes the rationale of treatment of an acute dislocation. Immobilization must be continued for a period long enough to permit repair of the lesion in

the capsule, else the stage will be set for recurrence of dislocation. As the problem of treatment of acute dislocation has been introduced and the importance of fixation (with the arm at the side, and the orearm at the front of the body) has been stressed, the age factor must be mentioned. *Under forty years of age* there is little danger of the development of the "stiff shoulder" syndrome through immobilization, but recurrent dislocation is a frequent problem and a very disabling one. *Over forty years of age* "stiff shoulder" syndrome is very apt to occur if the shoulder is immobilized, but the incidence of recurrent dislocation is very low. Therefore immobilization for acute dislocation of the shoulder should be practised only in the younger age group, the group in which dislocation is far more commonly seen.

SUBLUXATION MAY BE ELUSIVE

Furthermore, while considering the mechanism of injury in shoulder dislocations, we should differentiate clearly complete dislocation and subluxation. Complete dislocation rarely goes unrecognized. The patient cannot reduce it himself and function is restricted until medical aid is obtained, and the displacement reduced. Subluxation of the shoulder joint on the other hand, is a much more subtle diagnostic problem, and its recognition may easily be missed until it has become a recurrent problem. The humeral head displaces just over the rim of the glenoid, and is reduced spontaneously when the arm is brought down and forward. A clear history is usually given that the shoulder "goes out", and a snap or soft thud is experienced, together with relief of the sensation of displacement, when reduction occurs. The pathology in these cases is the same, very often detachment of the glenoid labrum from the bony rim of the glenoid, and if the acute injury is unrecognized and untreated, the stage is set for repeated incidents of subluxation.

ROTATOR CUFF TEARS

Tears of the rotator cuff may present a very constant story of the manner of injury also. The patient has a fall with the arm pinned at the side. The sudden strong contraction of the abductor muscles of the shoulder in attempting to raise the arm to a position of protection, meets an absolute resistance, and a tear of the musculo-tendinous cuff results. A tear may also occur when a heavy or unexpected load is taken on the partially adducted arm. In this injury, age is a very important predisposing factor. Degenerative change in the shoulder cuff of varying degrees is a constant manifestation of the aging process, and the resultant weakening of the intrinsic strength of the tendinous structure predisposes to tear. Tears are rare and unlikely before degenerative change appears, and thus are rare under forty years of age. They occur at a constant site in the tendon, just short of its insertion into the tuberosity, leaving a short stump of tendon attached to the tuberosity; and as the tendon of the supraspinatous is at the summit of the cuff, and takes the greatest load, it is torn most commonly.

Tears may be of varying degrees of severity. When complete rupture occurs, a definite dull snap may be felt or heard by the patient who then finds himself unable to elevate the arm. Tears of partial thickness may occur without definite history of an injury. The lesion is on the bursal aspect of the cuff, and presumably is produced by repetitive abduction activity. An occupation which requires repeated abduction of the arm

to an overhead position may produce this type of tear. *The fibres of the tendon appear to fracture much as a wire does when it is bent repeatedly at one point.*

THE SURGICAL NECK

A simple fall in which the weight of the body is taken on the arm is the common cause of fracture of the surgical neck of the humerus in an older person. Here again, age is the most important predisposing factor, and the same trauma to produce any injury at all.

DISLOCATION AND CUFF INJURY

Study of the mechanism of injury serves only to provide clues, and diagnosis depends finally upon the positive findings of physical examination and X-ray. A good clue however often leads to the rapid solution and understanding of a problem. There is some danger of oversimplification of diagnostic thinking by this process. One must, in particular, be aware of the possibility of multiple injuries to the shoulder. For example, inquiry into the mechanism of injury, and the findings on examination may indicate a dislocation of the shoulder, and X-rays too, may show no further injury (although a greater tuberosity fracture or other fracture is often revealed particularly in the older age group). The co-existance of a complete tear of the rotator cuff may be entirely concealed and unrecognized. Because of loss of function while the shoulder is dislocated a tear cannot be recognized at that time; it will not be recognized at the time of reduction if the patient is under an anaesthetic; and for a considerable time after that, loss of power to abduct the arm may be attributed to pain, so that the tear escapes detection until a late stage in treatment.

FRACTURE AND CUFF INJURY

Again, complete tears of the rotator cuff may be associated with fracture of the clavicle or with acromio-clavicular dislocation. If the manner of injury is a fall or a blow on the outer aspect of the shoulder which fractures the clavicle it may also evoke a sudden forceful attempt to abduct the arm which is pinned at the side, and a complete tear of the cuff occurs. Because the pain associated with the fracture of the clavicle prevents abduction or because the doctor's attention is distracted by his recognition of a fracture, the rupture of the cuff may easily be overlooked.

ELIMINATING THE PAIN FACTOR

Diagnosis of tear of the rotator cuff is indeed the greatest problem in diagnosis of shoulder injury. Complete rupture may be quite apparent because of the inability of the patient to initiate abduction, but partial tears may retain near normal power of abduction, and the chief presenting clinical symptom is pain on abduction. If pain is severe, abduction may be totally inhibited even though there is no tear in the cuff. Local infiltration with novocaine is then a valuable diagnostic aid as it removes the pain factor in function. Diagnosis of a complete tear may be made with fair certainty if inability to raise the arm persists after pain has been abolished. In some cases power to raise the arm has been maintained in spite of a complete tear of the cuff (though the power of abduction is diminished), so that diagnosis cannot be absolute unless a direct exploration of the shoulder is done. If a diagnosis of a complete tear can be made, open repair is indicated; but if diagnosis is uncertain or a partial lesion only is suspected, experience has shown that a conservative plan of treatment should be followed. The period of

disability may be long in spite of early physiotherapy and an aggressive programme to keep the shoulder mobile. Diodrast arthrography is of some help in diagnosis of problem cases. A tear of the cuff is indicated if the diodrast passes from the shoulder joint into the subdeltoid bursa. The size of the tear cannot be judged by this however, and in patients over fifty years of age, degenerative change in the cuff may have established a communication.

LEGG-PERTHES' DISEASE

P. J. NEELANDS, 5T5

LEGG-PERTHES' disease is an aseptic necrosis of the upper femoral epiphysis. Commonly used synonyms for this disorder include coxa plana, Legg-Calve-Perthes' Syndrome, osteochondritis deformans juvenilis, and pseudocoxalgia.

In 1910, Legg in Boston, Calve in France, and Perthes in Germany each described this syndrome. Prior to this, all hip disorders in children associated with pain and limping were considered tuberculous in origin, except for congenital deformities. However, Legg and Calve and Perthes presented a small series of cases without signs of joint inflammation and with minimal signs of muscular atrophy. X-rays were just coming into use, and each worker was able by their use to demonstrate an avascular necrosis of the upper femoral epiphysis as the cause of the disease. Subsequent investigators showed that this process was an aseptic one, although at first, organisms were cultured from the curettings of the epiphysis. These, however, later proved to be contaminants, so that the true cause of the aseptic necrosis is still not determined.

Aetiology

Certain predisposing factors are known. Trauma preceding the onset of necrosis is a common finding in the history. Heredity also plays a part, and in one family, twenty-eight cases have been traced over five generations. One case is on record at the Hospital for Sick Children with a family history of the disease for three generations.

Other causes which are mentioned include previous virus infection, hypothyroidism, and congenital abnormalities of upper epiphyseal centres. Still others believe that these changes in bone structure are secondary to synovitis, with resulting sclerosis of local vessels and the tissue changes attendant upon ischaemia. Probably all that is definite is that trauma and heredity play important roles in aetiology of this disease, and of the two factors, trauma is the precipitating one.

Incidence

Legg-Perthes' disease occurs predominantly in children between the ages of two and thirteen years. The heavy concentration of cases is in the age range of four to nine years. The disease occurs in males four times as commonly as in females.

Pathology

The disease is bilateral in ten to twenty per cent of cases. The upper femoral epiphysis undergoes necrosis due to occlusion of its blood supply.

The neighbouring metaphyseal bone becomes hyperaemic and osteoporotic. Regeneration and resorption of bone occur from the epiphyseal line, and these two processes occur simultaneously. During the period of reconstruction, the femoral head and neck are softened and weight-bearing can cause flattening of the femoral head and a thickening of the neck. These changes are irreversible.

Clinical Picture

An otherwise healthy youngster presents with a limp, with or without pain in the hip and knee.. This limp cannot be explained by any evident local lesion. Upon examination it is found that internal and external rotation at the hip joint are limited. Abduction is also restricted. The tuberculin test is usually negative.

Diagnosis

Early on, X-rays of the hip will show in A-P and lateral views an increased density in the upper femoral epiphysis and an increased radiolucency beneath the articular cartilage are seen. The lateral portion of the epiphysis may be eroded, and the joint space shows increased depth. Osteoporotic changes may be present in the metaphysis. Later X-rays show varying amounts of fragmentation. (See figure 1.) If no treatment has been undertaken the femoral head will appear flattened.

Course

The course depends upon the stage at which treatment is begun. Early effective treatment can shorten the course to 26–32 months. Without treatment, Legg-Perthes' disease may last three years or more. The full course is run when the epiphyseal bone has been completely restored, as seen radiographically.

Treatment

The principle to be followed is the avoidance of weight-bearing until the femoral epiphysis has regenerated sufficiently to bear the body weight without flattening.

At the Hospital for Sick Children, the preferred device for achievement of this principle is the Taylor walking brace. This apparatus suspends the limb free in a kind of caliper, so that the body weight is transmitted to the ischial tuberosity by two straps which are attached to a metal band about the waist. Adjustments can be made every three months or so to allow for growth. At each visit, the caliper can be lengthened and the child's progress and degree of cooperation can be assessed.

Figure 1

Figure 2

The advantages of the walking brace are many. The expense of treatment is much less than for long hospitalization. The child is happier because his activities are not so severely restricted as might be at first supposed.

Other forms of therapy exist, but are for various reasons held to be generally less desirable. Complete bed rest may be employed but is hard to enforce. Recumbency may be maintained by use of the Wu splint of Pike and Goff which permits moderate activity but prevents normal play. The use of a plaster spica is not now favoured. In bilateral cases, one may well be obliged to employ long periods of bed rest, and this is held to be the treatment of choice in such cases.

In seventy-five cases of Legg-Perthes disease at the Hospital for Sick Children in Toronto, results of the use of Taylor walking calipers were as follows:

Excellent results:	32 cases
Good results:	25 cases
Fair results:	9 cases
Poor results:	2 cases

The criterion for efficacy of treatment is closeness with which the upper femoral epiphysis and the femoral neck approach the normal in appearance. A rough guide to success is thus, how much real shortening is present in the extremity.

In the series reported here, no allowance has been made for such factors as late diagnosis, late treatment or poor cooperation on the part of the patient.

Prognosis

Prognosis can be correlated with the degree to which the femoral epiphysis approaches the normal. Flattening is irreversible, and leads to traumatic arthritis in middle age.

Summary

Legg-Perthes' disease is reviewed, and the accepted treatment in this centre is briefly discussed.

It is emphasized that a child between two and thirteen years complaining only of pain in the hip, and a limp may well be suffering from this disease. Examination of the patient should include estimation of function of hip joints and X-ray studies of these joints in A-P and lateral views. With earlier diagnosis, the course of Legg-Perthes disease can be shortened and the ultimate residual damage minimized.

Acknowledgement

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POST-TRAUMATIC REHABILITATION

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POST traumatic rehabilitation is a broad subject which can be, and has been, discussed at great length from a variety of viewpoints. It is unfortunate that the ultimate goal of restored function is sometimes lost in a smoke-screen of controversial operative techniques and radical theories.

It is immaterial which member of the rehabilitation team contributes the most, or by which method he does so. The physician, surgeon, psychologist, social worker, physical and occupational therapist are all working toward a common goal. The patient and his community are partially interested in maximum rehabilitation after trauma and should be guaranteed the right to utilize the medical profession and its allied services as a co-ordinated means to that end.

The purpose of this article is to indicate briefly some of the contributions which physical and occupational therapy can make toward the total rehabilitation of the injured patient. The two therapies are primarily concerned with the physical and psychological rehabilitation phase of treatment and should receive careful consideration from those planning an integrated programme.

Occupational Therapy

The benefits of treatment by means of participation in occupations and remedial activities can be extended both to the psychological and to the physical aspects of a patient's disability. This overall utilization of the potentialities and resources of the patient's own mind and body makes occupational therapy a valuable treatment in many types of psychiatric and medical conditions. The Workmen's Compensation Board Rehabilitation Centre, at Malton, Ontario, and the hospitals administered by the Department of Veterans' Affairs are sites where the value of remedial occupations has been proven in the treatment and assessment of traumatic conditions.

Speech therapy, and training in the use of prostheses, adapted equipment, and self-help devices, as well as assessment of work tolerance are specialized fields in which the occupational therapist can be of great assistance. Patients who have sustained brain or spinal cord lesions are trained to perform the activities of daily living and to become self-sufficient if possible. Manual skills are only one medium of treatment. For certain patients their psychological value is important, but in other cases such tasks can be used to evaluate skills and to determine capabilities prior to training and job-placement.

Recently, more and more emphasis has been placed upon the consideration of the patient as a person, rather than as a disease state or condition. Psychosomatic researches have brought home the fact that the patient's mental attitude to his illness has great bearing upon the course of his recovery. Social, economic, and emotional problems definitely influence the rate of recovery, as well as the adjustment to the disability, and even the degree of ultimate rehabilitation.

Because the field of occupational therapy extends from the early stages of hospitalization into the reconditioning period, the occupational therapist must

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have a sound basic knowledge of anatomy, kinaesiology, physiology, psychology, and of physical and psychiatric conditions. She must be trained to evaluate the progress and capabilities of the patient in the occupational situation, to adapt her media of treatment to develop neuro-muscular patterns of co-ordination, and to increase joint range and muscle power. She must be able to up-grade a work tolerance programme and to estimate the requirements of a job and relate them to the degree of rehabilitation achieved. In addition, the occupational therapist must know and utilize the social services, vocational guidance centres, and facilities for sheltered employment available to her patient.

SAMPLE CASE

Patient Male, age 40.

Injury Fracture to right carpal scaphoid.
Occupation Master electrician.
Degree of Rehabilitation Desired Return in full to former employment.
Comments The occupation of this patient demands a high degree of co-ordination and muscle strength in the right upper extremity. A strong grasp and dexterity are important in the right hand for the skillful use and manipulation of tools.
Occupational Therapy During the period of immobilization of the right wrist, the patient is encouraged to work with a wood chisel, so that the strong finger flexion causes the synergistic contraction of wrist extensors. Braid-weaving is done in a modified position to encourage strong finger extension and synergistic contraction of wrist flexors.

After the plaster has been removed metal work is introduced as an activity which can be progressively graded for strength and co-ordination. The use of a planishing hammer to encourage mobilization of the wrist joint, and the handling of tools and materials similar to those of the patient's trade, are major factors in the choice of this specific occupation for this patient.

Rationale During immobilization, the strong contraction of the forearm muscles prevent a great deal of muscle atrophy without causing movement at the fracture site. Gentle half-range movements of the fingers would be useless as the synergistic contractions of the forearm muscles would not occur. Local circulation is increased by muscle contraction.

In each of the activities used in his occupational treatment, the patient is using his entire arm and hand in a functional cally and in the maintenance of neuro- pattern that is important both psychologi- muscular co-ordination. The free joints above and below the plaster cast are being exercised through a functional range, and muscle action is not allowed to deteriorate through disuse.

Interest is readily maintained by the use of constructive and stimulating occupations.

Physical Therapy

The first case of post-traumatic rehabilitation incorporating physical therapy was recorded in 3000 B.C. Heat, massage, and exercise have been used therapeutically since pre-historic times, and have not yet been discredited as useful treatment media.

In the last century, enthusiastic masseuses almost succeeded in obscuring the benefits of intelligently used remedial exercises, and *some people still confuse massage and physical therapy* as one and the same thing. Massage is used by physical therapists to relax muscular spasm, to stimulate circulation, to relieve congestion caused by oedema, and to relax a tense, anxious patient. Gentle lubricating massage to scar tissue or indurated skin is useful as a softening procedure.

The physical therapist applies her knowledge of anatomy, physiology, and kinaesiology to the scientific re-education of specific joint and muscle functions and to the general co-ordination of

movement. An intelligent patient can be instructed to do his remedial excercises without supervision, but the results are often disappointing. The average layman has no criteria for judging the degree of movement possible at a given stage of recovery. He has no conception of which movements will endanger healing tissues or which movements are possible and important to an ultimate maximum recovery of function. Pain and lack of understanding lead to substitution of one muscle group or joint movement for another. Inbalance of muscle power and loss of joint range lead to crippling deformities and permanent disabilities in many cases.

At the one extreme we have the patient who considers his disability a personal affront and persists in forcing the injured part to do too much. At the other end of the scale, we have, for instance, the patient with a fractured ulna who treats his injury with such exaggerated respect that he completely inhibits movement and acquires a frozen shoulder for all his trouble.

Intelligent patients require a minimum of instruction and supervision, but they are definitely entitled to that minimum. Most patients, however, lack the initiative and the persistence necessary to supervise their own treatment regime, and those who respond only to coaxing, cajoling, or to authoritative firmness, are the persons who require the most intensive physiotherapy. Proper neuromuscular re-education following spinal cord, brain, and peripheral nerve lesions is always an extensive and prolonged business.

Physiotherapists receive a specialized training, and with it are cognizant of pathological signs and symptoms which may present themselves during the period of rehabilitation. They are taught to treat joints and tissues with respect, and to restore function carefully and gradually. Forced movements are never used where tissue damage might occur, and fracture lines are not disturbed during the process of union.

Physical therapists work by the doctor's prescription, and are under his guidance. Therefor, it is the doctor's responsibility to prescribe the treatment that is required and really to supervise in a constructive fashion the patient's handling under the therapist.

The *general aims* of physiotherapy in almost every post-traumatic condition are as follows:

1. Relieve pain.
2. Improve the circulation of blood and lymph.
3. Prevent or reduce deformities.
4. Prevent muscle atrophy, loss of muscle power, and to strengthen weakened musculature.
5. Maintain and/or re-educate kinaesthetic sense.
6. Maintain and/or re-educate neuromuscular co-ordination. i.e. to restore skilled movements.
7. Contribute to the general well-being of the patient.

The *methods employed* include remedial and gymnastic exercise aided by massage, heat, electrical stimulation, ultra violet radiation and hydrotherapy.

MASSAGE Massage has been mentioned earlier. It is used to relieve pain by assisting circulation and by relieving muscle spasm, and to relax a tense patient. Special types of massage are frequently beneficial in specific treatment situations.

HEAT The sources of heat include short and long wave diathermy, infra red generators and constant electrical currents.

The source and the heat produced is modified to suit the condition treated. Acute inflammatory reactions are treated with frequent, short, non-penetrating

doses below the threshold of heat sensation. This type of heat does not cause an undesirable hyperaemia. Contracted, fibrosed joint structures can be "softened" by deep penetrating doses of heat which cause a great increase in local circulation. The relaxation of muscle spasm and the "softening" of proliferated fibrous tissue have not yet been satisfactorily explained, but do occur and are important adjuncts to treatment. Massage, heat and exercise are usually given in order that the maximum amount of benefit may be derived from each modality.

ELECTRICAL STIMULATION Electrical stimulation is used upon muscles or nerves to re-educate muscle contraction or to test neuro-muscular function. New square-wave type currents have been developed recently and have greatly increased the value of muscle-nerve tests.

Constant current treatments, and superimposed galvanic and faradic current treatments, should be mentioned for their value in specific situations. Each type of current has distinct properties which may be utilized for specialized purposes.

ULTRA VIOLET RADIATION The benefits of ultra violet radiation are well known but rarely are incorporated into post traumatic treatment. One of the most important justifications of its use is the increase in calcium and phosphorus deposition in bones necessary for union of fragments following fractures.

If infection or ulceration are present, the destruction of surface bacteria and the improved circulation of the skin following ultra violet irradiation can be very useful. Destruction of superficial epidermal cells by the action of ultra violet rays results in peeling and the eventual stimulation of growth and replacement.

The increase in general well-being following ultra violet treatments is well known and could be used with benefit in many cases.

HYDROTHERAPY This is a treatment medium offering so many specific advantages that it has become a separate modality. The principle of buoyancy and weightless exercise can be paralleled in other forms of remedial exercise. In hydrotherapy, the psychological benefit of swimming or floating despite a handicap, or of seeing a movement he did not believe existed, is of paramount importance to a patient. The use of increased temperatures to relieve pain, to stimulate circulation, to produce relaxation or stimulation, earns benefits which are both physical and psychological.

REMEDIAL AND GYMNASTIC EXERCISES

Remedial exercises are usually specific for a muscle or muscle group. Gymnastics are used for general conditioning and are particularly useful in the later stages of rehabilitation. Body mechanics and the principles of leverage, pulleys, springs, and suspension are "tricks of the trade" which the physiotherapist utilizes to gain the correct type and degree of muscle contraction. Correct breathing and conscious relaxation are techniques which can be taught by the physiotherapist. In cases of trauma to some part of the thorax, as with stab wounds, instructions in breathing techniques may be given the patient so that he can increase his vital capacity and restore as much as possible the function of his respiratory system.

The ability to relax muscles under tension, to conserve muscle function for the maximum efficiency in movement and in the maintenance of posture, and the ability to carry on respiration efficiently are all valuable to any patient, especially if he must remain for long

periods upon a restricted activity programme.

An amazing number of patients exhibit chronic fatigue, insomnia, and inefficient circulation due to neglect of these principles, and while the symptoms are not attributable in every case to poor posture, nervous tension, and apical breathing, there is often an evident correlation.

Summary

I have attempted briefly to indicate a few of the therapeutic principles employed by physical and occupational therapists. The use of these principles in post-traumatic rehabilitation encourages both a shorter period of disability and a maximum return of function. An intelligent use of the two therapies is essential to a full rehabilitation programme, and this implies a knowledge of their limitations as well as the possibilities of their use. While the therapists are not qualified to diagnose disease or to prescribe treatment, they are certainly trained to make the most of the procedures for treatment at their command, to the extent indicated in a given case. The success of physical and occupational therapy in rehabilitation depends upon, first, the awareness of the physician or surgeon of the treatment media at his disposal, and, secondly, upon the professional knowledge and skill of the therapist carrying out his instructions.

The increasing emphasis being placed upon restoration of total function in post-traumatic rehabilitation indicates the growing necessity for a close alliance between the physician and the therapist for better integration of effort toward this common goal. A better realization on the part of all members of the medical profession of the important role to be played by the physical and occupational therapists in the handling of post-traumatic cases and in many other situations as well can only serve to improve the standards of treatment and to improve the chances of full recovery of the patient, who is, after all, the reason for existence of both professional services.

WHAT A MEDICAL STUDENT SHOULD KNOW ABOUT PERFORATED DUODENAL ULCER

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ACUTE perforation is one of the more common complications of duodenal ulcer. The part it plays in the differential diagnosis of the acute abdomen is a major one, and for this reason it is important to have more than a nodding acquaintance with the signs and symptoms of this lesion.

The typical story of perforation is well known to all. It usually follows many years of seasonal annoyance from post-prandial epigastric distress. Not infrequently an exacerbation of this discomfort occurs for a week or two prior to perforation. In roughly fifteen per cent of cases the blowout occurs with no previous upset in gastrointestinal physiology. The sudden onset of severe epigastric distress accompanied or followed by right shoulder tip pain are sheet anchors in the diagnosis. Occasionally the epigastric discomfort is relatively minor, and acute right lower quadrant

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pain and tenderness are severe enough to warrant the diagnosis of acute appendicitis. In some cases the patient is prostrated by the severity of the pain, and lies immobile in bed. Others, quite contrary to accepted belief, prefer to sit up in bed, or thrash around, while still others wander about, moaning and groaning, pressing the upper abdomen with both hands. Very rarely there are periodic exacerbations which can be confused with crampy pain, and when associated with incessant nausea and vomiting, suggest a high small bowel obstruction. Sometimes the pain, of whatever nature it may be, follows a heavy meal of either food or alcoholic beverages. The surgical cliché that perforated ulcers never bleed and bleeding ulcers never perforate still holds true for the most part, and very rarely is perforation accompanied by haematemesis. If the rule is transgressed, it can usually be explained by the presence of coincident anterior and posterior wall ulcers, both of which have taken part in the production of symptoms.

The importance of physical examination cannot be stressed too much. The findings vary greatly with the time interval between perforation and examination, and the amount of peritoneal soiling. In the typical cases seen within a few hours of perforation the patient may be pale, cold, clammy, and in a state of shock, with subnormal temperature and extremely low blood pressure. The abdomen presents a boardlike rigidity with hyperaesthesia, tenderness and rebound tenderness. The findings are most marked in the upper abdomen, and may trail off into the lower abdomen. If the leak is less massive the patient may not appear acutely ill. The temperature and blood pressure are within normal limits. Increased resistance, tenderness and rebound tenderness often are confined solely to the epigastric region. In some cases, where the gastric content has dribbled down the right gutter, maximal findings of a similar character may be limited to the right lower quadrant. Rectal tenderness in all these instances may or may not be present. In only fifty per cent of cases in our series was free air found under the right diaphragm, either on physical examination or X-ray screening. The absence of free air, therefore, does not influence the diagnosis of acute perforation. In all cases in this series, leucocytosis was noted at the time of examination. It varied from 9,000 to 25,000 cells. Urine findings are usually non-contributory.

It is well to have knowledge of the course a patient suffering from perforation might follow as the hours go by. After eight or ten hours have elapsed from the time of perforation, the individual may pass into a state of apparent improvement. The boardlike rigidity may seen to decrease and all the findings of the intraperitoneal catastrophe lessen. It is in this interval that the careless observer might be lulled into a sense of false security, and decide that very little is wrong with the patient. This is the treacherous period just preceding a widespread bacterial peritonitis, and during which diagnosis may be so difficult. Usually, however, if enough care is taken with examination, the correct answer is forthcoming. Often there is elevation of the temperature, significant elevation of the pulse, and definite signs of peritoneal irritation in the upper abdomen. This seemingly quiescent period lasts for an indefinite time, and is then followed by the signs and symptoms of a widespread generalized peritonitis.

The different diagnosis of acute perforation must include the common lesions which involve the upper abdominal organs and less commonly two extraperitoneal structures. Perforated gastric ulcer, acute cholecystitis, acute pencrea-

titis, acute appendicitis and acute small bowel obstruction are all in the picture. Coronary thrombosis and inflammatory lesions affecting the diaphragmatic pleura on the right side are occasionally encountered. In our series of one hundred and fourteen cases of acute perforation of a duodenal ulcer, only five patients were members of the fairer sex, whereas the majority of those suffering from acute cholecystitis were female. These findings may be of minor help in the differential diagnosis. Occasionally the serum amylase determination may be a deciding factor for or against perforation as opposed to pancreatitis.

Once the definite diagnosis of a perforated ulcer has been made, two courses of *treatment* are open for consideration: operative and non-operative. In the former category two procedures are available. In some centres, its treatment consists of immediate partial gastrectomy. This may be performed in cases where operation is undertaken early, where soiling of the peritoneal cavity is minimal and the patient is in excellent condition. In this centre, however, operation consists of the less radical procedure of visualization of the ulcer and its closure with an attached or detached omental graft gently held in position by three catgut sutures. The method is easy, rapid and efficacious. Prior to any surgical interference the patient is prepared by insertion of a duodenal tube for continous decompression of the stomach. An attempt is made to balance the fluid and electrolyte loss by giving suitable intravenous fluids. In some instances, blood transfusion may be necessary to alleviate the accompanying shock and get the patient in proper condition for operation. It is perhaps a wise precaution to commence the use of antibiotics such as Dicrysticin in the prevention of serious bacterial peritonitis post-operatively.

The non-operative treatment consists of continous gastric decompression by means of an indwelling duodenal tube and suction, intravenous feeding, sedation and antibiotics. The reasons for conservative treatment as followed by the proponents of this method are mainly three in number—*First*: The results of this treatment in many hands closely approach those of surgical interference. *Second*: Not infrequently at the time of operation the perforation is found to be closed by fibrin, omentum, or covered securely by the liver, and operation is believed unnecessary. *Third*: Many patients who refused operation or who were not operated upon because of a concurrent chest or cardiovascular complication, recovered on this treatment. The pros and cons for either radical or conservative treatment will not be discussed here. Suffice it to say that there are several excellent methods of dealing with this very important lesion.

In our series of one hundred and fourteen cases of perforation of duodenal ulcer, treated surgically by simple closure, the mortality rate was 6.3 per cent. In only two cases was intraperitoneal suppuration the cause of death. In the other five cases, pneumonia, pulmonary embolism and uraemia were the precipitating factors. Two cases refused operation and died.

We were able to follow seventy-three patients of the series for five to twenty years after perforation. We found several interesting results at the time of follow-up:

25% were asymptomatic.
8% had only occasional mild symptoms suggestive of further activity.
22% had typical postprandial epigastric pain in seasonal attacks.
12% had severe ulcer pain with symptoms of penetration, bleeding or obstruction.
35% had had another operation at the time of follow-up.

By grouping these figures we arrived at an easy method of remembering the results:

One-third have no trouble
One-third have trouble
One-third have *bad* trouble.

Our follow-up suggested that a certain number of patients suffering from duodenal ulcer which had perforated on at least one occasion, can get along perfectly well with no restrictions on diet, smoking or drinking. We still feel that a fairly rigid medical therapy is important in attempting to control these patients. It is true, however, that the majority do not, cannot or will not follow the medical advice given to them, and will stray from their diets and will continue to smoke and drink.

Our follow-up showed, too, that one cannot predict with any degree of assurance at the time of original perforation or in the six months following operation whether any one specific patient will or will not have trouble with his ulcer in the future. Some patients continued to have symptoms for two years post-operatively and when advised to undergo gastrectomy, refused. Many of these were well at the time of follow-up ten to fifteen years later. On the other hand, some who were free from pain and ulcer distress for years after perforation developed further symptoms five to ten years later and underwent further operative procedures. Every case therefore must be treated on its individual merits.

It is interesting to note that six per cent of this group subsequently had another perforation. Of the remainder undergoing further operation, all but one required surgery for a coincident or subsequent posterior wall ulcer, rather than the original anterior wall ulcer.

Summary

1. Perforated duodenal ulcers, occurring most frequently in males, are a common cause of the so-called "acute abdomen."

2. Most perforations follow a previous history suggestive of ulcer.

3. Fifteen per cent of perforations occur with no antecedent ulcer story.

4. The typical signs and symptoms are discussed. The "treacherous period" should be noted with care.

5. The usual treatment consists of simple closure of the perforation with omental patch. Other forms of therapy are available.

6. The mortality rate in a series of one hundred and fourteen cases is 6.3 per cent.

7. Six per cent of perforated ulcers re-perforate.

8. Adequate medical treatment should be carried out post-operatively.

9. Roughly one-third of ulcer patients who perforate will have very little or no trouble. One third will have further typical or severe symptoms. One-third will require further operation.

The Use of Cancellous Bone Grafts in the Treatment of Subacute and Chronic Osteomyelitis: J. W. Hazlett, (Orthopedic Service, Sunnybrook Hospital, Toronto). The Journal of Bone and Joint Surgery, November 1954, pp. 584–590.

One hundred and one cases of cancellous chip bone grafting operations for filling of infected bone defects are reviewed. They include ninety compound fractures, six simple fractures infected after open reduction and five hematogenous osteomyelitis with abscess. All patients had radiographs taken and preoperative culture of wound discharge. Penicillin was administered routinely in doses 20,000 U q3h for 2 days preoperatively and seven days postoperatively. Single-stage sequestrectomy with cancellous bone chips mixed with penicillin and sulphathiazole powder was done in seventy-six of the 101 cases. In the other twenty-five cases sequestrectomy and packing of the wound were performed one to eight days before the bone grafting operation. In fourteen instances full-thickness skin graft was prepared before the bone grafting operation. In another eight cases local skin graft or rotation flap was done at the time of bone grafting.

Short term follow-up showed primary or delayed primary healing in 87 per cent of cases. Eleven more cases achieved secondary healing with further operation and in three cases no healing was obtained. Observation after five years revealed a recurrence rate of 20%. These recurrences were successfully overcome by minor procedure except in five patients who suffer from repeated exacerbation of infection.

At present 91 per cent of the lesions are satisfactorily healed with a partial or complete bone graft intact. There have been 10 failures.

The criteria to be fulfilled for a successful cancellous chip bone graft in osteomyelitis are:

1. Control of infecting bacteria by antibiotics systemically or locally.
2. Full-thickness skin coverage of the wound.
3. Complete removal of all infected bone and soft tissue.
4. Sufficient saucerisation of bone and excision of soft tissue scar to expose a vascular bed for bone grafts.
5. Decortication of the chip grafts.
6. Obliteration of all dead space by the loose packing of sufficient small bone chips.
7. Immobilization of the affected area to allow soft tissue healing or bony union of an associated fracture.

I. G. L. Van Praagh

Trigger Finger in Adults and Children: John J. Fahey and John A. Bollinger; Journal of Bone and Joint Surgery, Dec. 1954, pp. 1200–1218.

Stenosing tenovaginitis is a constriction of a tendon sheath, usually associated with an incongruous tendon surface and results in interference with the normal smooth gliding of the tendon within its sheath. When the flexor tendon sheaths and tendons of the fingers are involved, snapping or triggering may occur as a result of the sudden blockage of the tendon prominence against the stenosed sheath and the abrupt release following the increased force required to pass the obstruction. The author reviews the findings in twelve cases of stenosing tenovaginitis of thumbs in children, thirty-one cases in adults and

twenty-eight cases with stenosing tenovaginitis of the second, third, fourth and fifth fingers.

The occurrence of trigger finger in twins, the appearance of the syndrome at birth or early childhood and the frequent bilateral involvement all lend support to the theory that children may have congenital predisposition to this condition.

The most frequent presenting deformity in children was locking in flexion and in adult, snapping was the most common complaint. Loss of active flexion, except for a few degrees with the and in adults, snapping was the most common presenting deformity in adults.

In the adult, digital fibrous-sheath involvement is prominent and tendon changes are minimal, as compared with the condition in children. Palpable mass in the base of the thumb in children is frequently due to tendon proliferation and degeneration, resulting in a nodular formation.

Conservative treatment should be tried in adults seen shortly after the onset of symptoms. For adult patients who do not respond to conservative treatment and who are seen late, operative treatment is recommended. In children, only operative treatment has been satisfactory. Satisfactory results follow local excision of the tendon sheath, suture of the skin and encouragement of early motion.

I. G. L. Van Praagh

Fanconi's Syndrome, Treatment by Massive Dosage of Calciferol and Two-Stage Osteotomy: Martin Singer, Journal of Bone and Joint Surgery, Nov. 1954, pp. 633–636.

The author discusses Dent's classification of various "resistant" rachitic and osteomalacic conditions into the following groups: (1) Vitamin D and calcium lack. (2) The steatorrheas. (3) Bile salt deficiency: ? hepatic rickets. (4) Renal glomerular defect (polycystic kidney and other gross renal defects). (5) Renal tubular defect, uterocolostomy.

In *renal glomerular rickets* which is the classical "renal rickets" both glomeruli and tubules are damaged or destroyed leading to retention of urea, phosphates, etc.

In *renal tubular rickets* there is an inherited or acquired defect in tubular reabsorption. The group may be classified depending upon the site and extent of the tubular reabsorption defect resulting in:

1. Low blood phosphorous.
2. Glycosuria. These defects have been described as "resistant rickets" or Milkman's Syndrome.
3. Aminoaciduria including cystinuria.
4. Defect in phosphate, glucose and aminoacid reabsorption with a faulty urine acidification giving a low carbon dioxide combining power. Types (3) and (4) have been termed Fanconi's Syndrome, cystine rickets, and renal rickets.
5. Poor phosphate absorption with faulty urine acidification and high urinary calcium content.
6. Phosphate with faulty urine acidification, defective ammonia formation and high urinary calcium content. The differential diagnosis rests with the biochemist rather than the clinician.

The author reports a case which accords with Fanconi's Syndrome (types 3 or 4 of the classification), the signs and symptoms being due to defect in reabsorption affecting primarily the proximal tubules of the kidneys and involving amino acids, sugar and phosphates. Severe cases show dwarfism, gross rickets, hypophosphatemia, aminoaci-

duria including cystinuria, glycosuria, deposition of cystine in the body tissues, and acidosis. In this 16 month old boy with Fanconi's Syndrome and renal tubular dysfunction, rachitic lesions healed after massive doses of calciferol. There was simultaneous improvement in the biochemical state including disappearance of aminoaciduria. Two stage osteotomies were performed to correct severe bowing of tibiae. There were no toxic symptoms after 1 year of calciferol therapy in doses of 20,000 to 500,000 units per day.

I. G. L. Van Praagh

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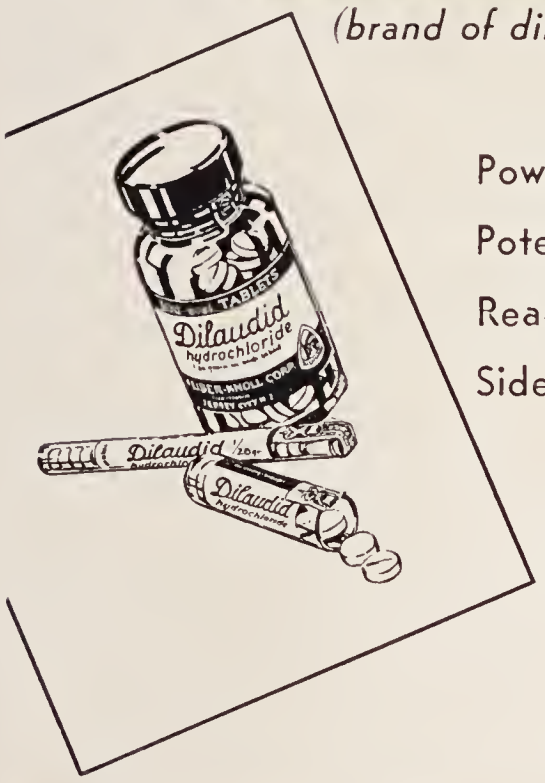

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*Pratt, R., & Dufrenoy, J.: Texas Rep. Biol. & Med. *12*:145, 1954.

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